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Nov 1879

Pulse Diastolism

Thesis, for the degree of, Doctor of Medicine,
presented to the University of Glasgow, October,
1879, by Wm. Jas. Fleming, M.B.-F. F.P.S.

Despite the enormous amount of labour and thought bestowed upon attempts to explain "pulse diastolism", much doubt still exists as to the manner of its causation. It is well known that waves can be produced ^{in elastic tubes} exactly similar to those existing in the arteries, and practically identical tracings can be obtained from both. It seemed to the author that, by taking tracings from india rubber tubes, under many different sets of conditions, without at first attempting to prove anything, facts might be elicited which would suggest a solution of the problem. The investigation thus entered upon, first led to a refutation of the different theories now held by physiologists. These we may now ~~now~~

advantageously enumerate

Views held ~~formerly~~

It may probably be taken for proved that the diastolic notch is produced by a wave proceeding from the centre to the periphery, as it has been abundantly shown that it occurs later, the further from the heart the observation is made. All recent authors have discarded the idea of a reflexion from the periphery.

There remain then five theories of which we require to take account

- 1 Vibration caused by closure of the aortic valves.
- 2 Vibration of the walls of the artery. (McVail)
- 3 A reflexion, from aortic valves, of a reflux of blood, produced by the resistance of the blood already in the more peripheral parts of the circulation.

(Galien)

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4 A second contraction, of, the root of the aorta,
following a distension, produced by a —
rebound from the great arteries in front.

(Burdon Sanderson)

5) An active contraction behind each pulse wave —
a vermicular contraction.

(C. S. Roy)

Methods employed

To represent the heart. First, Burdon Sanderson's
Schema with a head of water of about 1½ metres X

2nd A simple head of water of about the same
height

3^d A Higginson's syringe worked by the hand.
For artery I used various lengths of, extremely thin india
rubber tube made specially for the purpose — diameter
of lumen ⁵ millimeter.

X See Laboratory Hand book p 22155 40 Plate LXXXVI
Fig 211

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The amount of peripheral resistance was regulated, by (A) partial closure of exit tube, (B) elevation or depression of end of tube, and (C) by causing the water to escape into a vessel by an aperture near the bottom, and to overflow by an opening some inches higher; this vessel being raised and lowered. (See Figure V)

By this last device a resistance, closely analogous to that offered by the mass of blood in the capillaries, is obtained and can easily be altered in amount, by elevation, and depression of, the vessel. Indeed this simple arrangement seems much superior to constricting the aperture of exit as is usually done.

The movements of the artery were recorded by means of my *Tambour Sphygmograph*, described in the *Journal of Anatomy and Physiology*. This is simply a *tambour* with a wire fixed to its centre, perpendicular to the membrane, armed at the end with a button for application to the artery or tube. Above the button can be placed split weights. From this the motion is communicated to the recording *tambour*, and traced on the cylinder as usual. Very numerous modifications of these methods were employed and a few of them are and a few of them are described when required in the subsequent portions of this paper.

Arguments against the views at present held

First Theory - Vibration caused by closure of, aortic valves.

The schema was arranged in the ordinary way, but, immediately beyond it, a T tube was introduced, and the free leg connected with ²mercurial manometer (Fig M) of considerable calibre. The proximal end was thus put in communication with the part of the experimental tube representing the aorta, and was filled with water above the mercury. The distal was closed, air being above the mercury. The sphygmograph was applied about 1 metro from the schema. On working the instrument slowly, tracing 2 was obtained. In this the dicrotic rise is well marked, and also the vibration caused by the closure of the aortic valve, occurring long after. ^{air in tube} The closure of the aortic valves cannot then be the cause of the secondary wave, although, in accordance with the theory to be advanced, it will necessarily occur almost synchronously with the origin, at these valves of the wave. Dr. McTear has shown that dicrotism may be produced by a simple depression of a piston, and argues from this, that the aortic valves do not produce the wave, but it seems evident that the cessation of the descent of the piston most perfectly represents the closure

of the valves, and that therefore the experiment rather goes to prove the opposite of the conclusion he draws from it. In the tracing (2), the diastolic notch must have been produced independently of any action of the valve representing the aortic, as, from the construction of the Schema, the opening of this valve, and the closure of the Auriculo-ventricular, are simultaneous, and coincide with the ^{beginning} of the systole, so that at the moment of the secondary rise in Trace 2 the instrument was at rest, the aortic valve open, the auriculo-ventricular shut.

Second Theory - Vibration of the walls of the artery

This theory, as far as I can trace, was first distinctly advanced by Dr. McVail, in the paper just referred to. It seems inherently improbable, from the 'damping' effect the surrounding tissues must have upon the vessel walls. An artery differs from an ordinary experimental elastic tube, in being tightly packed among the tissues, not surrounded by air, and any vibration of its walls would be damped, if not extinguished by the investing structures. To show that this is the case the following method was resorted to. The button of the sphygmograph was cemented to the wall of the tube, no weight being put upon it, and the membrane of the tambour being very slack. Next the tube and Sphygmograph button were immersed in mercury,

to such a depth that only the wire, a thin one, —
emerged from the liquid. By this arrangement the
chances of the momentum of the instrument affecting
the traces are practically eliminated, and the disposition
of the apparatus must have closely simulated the
condition of the vessel, packed amongst the tissues.
The simple tube, opened and closed with the finger,
was employed; the manometer being introduced as
above described. The tracing obtained is figured in
No 4, shewing that a marked diastolic rise may exist
under circumstances which preclude the possibility of
vibration of the wall of the vessel itself; This experiment
seems also conclusive that the theory attributing the
notch to vibration of the sphygmograph is untenable
but this has practically already been disposed.

Theory 3 Reflexion of a ~~wave~~^{wave} produced by
peripheral resistance

Galabin

This theory is consistent with my observations in so
far as it attributed the phenomenon to a wave
reflected from the cardiac end of the aorta, but
the explanation of the method by which the

original wave is caused to travel towards the heart. does not seem satisfactory. It must be admitted that a closure of the cardiac end of the aorta must exist, or the circulation could not go on, and this closure may either be, as in health, the shutting of the aortic valves, or as in mitral insufficiency the closed, or even partially filled ventricle. But the peripheral resistance is more yielding than this, and it is difficult to conceive how it could produce a reflected wave of sufficient intensity to cause, after being again reflected from the cardiac end of the aorta, a rise of the extent of the diastolic notch. Besides, as will be dwelt upon in the sequel, the diastolic notch increases with diminution of peripheral resistance, while by this theory we would expect it to increase.

Theor. 4. A second contraction of the aorta, after distension produced by rebound from peripheral vessels.

Burdon Sanderson

The objections to Dr. Gallen's theory are equally applicable to this, indeed, practically, if such a rebound exists, it must dilate the aorta, or, at least, arrest its contraction as well

as be reflected from the cardiac end.

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Theory 5 A vermicular contraction of the ~~pulse~~
artery behind each pulse wave

C. S. Roy

Dr. Roy's revised theory of a peristaltic contraction of the vessels seems at once negatived, by the case with which a dirotic rise can be produced, in elastic tubes which he surely cannot consider capable of an "active vermicular contraction" but, besides, in tracing we have a curve evidently representing the dirotic notch, and in the arrangement which produced this tracing the whole experimental tube, from the point at which the closure representing the valve was made to the end, was constructed of glass tubing joined by the shortest possible pieces of india rubber tube, except, at the one place, where the flexible pipe was about two inches long to permit the application of the sphygmograph. In this experiment there was only about three inches of the tube, in all, india rubber, and yet we have ~~the~~ a very marked ~~notch, probably the product of,~~
~~and~~ dirotic notch (a in trace)

Explanation offered

All the theories above enumerated having ~~having~~ failed to account for ^{the} phenomena, a consideration of a great mass

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of tracings and of the mechanical conditions involved led the author to attribute the second ^{and} rise to a rebound from the cardiac end of the aorta, of the wave produced, by the aortic contraction itself. To make this clearer, let us consider what happens if an elastic ball such as Higginson's syringe be compressed. We have here the fluid it contains, driven equally in both directions back and forward, so that two streams of fluid are produced travelling in ~~both~~ ^{opposite} directions, and if there were ~~no~~ ^{no} valves or other obstruction, equal quantities would flow from either exit tube but, with a ~~valve~~ preventing reflux through the inflow tube, we have the fluid which would have passed out by it in the absence of any ^{rebounding} obstruction, and the whole fluid passing out by the unobstructed orifice. The aorta is directly comparable to the ball of the syringe, and in speaking of the aorta in this connexion, we may probably include the first part of the great vessels arising from it. The ventricular systole distends them to the utmost limit, the greatest distension being just before its cessation. This systole ceases more or less abruptly, and the aorta instantly begins to contract upon its contents, not by a species of peristaltic action, but simultaneously, or nearly so, in its whole length. The blood is therefore driven back upon the aortic valves with the same force as it is driven forward into the circulation. This backward wave is universally admitted to close these valves,

and this ^{is} probably its first action, but having effected ^{it} thus, it must be reflected from them, and it is this reflexion which, in the author's opinion passing down the vessels, or rather acting on the mass of blood they contain, produces the dirotic wave. If this is the true explanation, we must consider how it can be demonstrated, and how it explains the conditions found to influence dirotism.

Experimental demonstration

If a quantity of fluid be injected into the ^{part of the} experimental tube representing the aorta, the injection beginning at the moment when distension ceases, an increase in the dirotic wave should be produced. — An increase if the dirotic notch is present without the injection; a production if previously absent. Many methods of producing this influse of fluid into the artificial aorta at the exact time were tried. Amongst others (a) a manometer introduced as described at p. 5 and as shown in the drawing. at M. A vertical tube of considerable length substituted for the manometer and closed at the end — the upper portion containing air. (c) A dilatation (Hogginsons syringe without valves) compressed by a flat board heavily loaded. By these devices the wave has been uniformly increased, or, if absent, produced. Of the first of these which gave the most marked results we have in the appended tracings the

following examples.

- 2 & 7 the manometer in } other conditions the same
 3 & 6 the manometer out }
 4 the manometer in Sphygmograph glued to tube
 and immersed in mercury

5 the manometer out otherwise same as 4

In these the effect of the injection of fluid produced by the manometer in increasing, the diastolic notch is well marked. And it is worth notice how the second ^{any} vibrations which are so apt to occur in experiments with elastic tubes are wanting, when the conditions favouring vibration in the tube are removed by its immersion in mercury, and a state of matters closely analogous to that existing in the arteries is thereby produced. In 8 & 9 we have tracings taken from an arrangement of glass tubes, connected by the shortest possible elastic joints, except at one point, where about two inches of india rubber tube was interpolated, to permit of the application of the Sphygmograph; otherwise the arrangement is the same as with the elastic tubing; in 9, the manometer being used. In these curves the diastolic notch is represented at (a), and the enormous increase of this curve when the manometer is introduced, compared with the same curve figure 8 without it, seems only explicable by the theory above propounded. Indeed if tracing 8 be compared

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with tracing 10 in which the bottom of the sphygmograph was rapidly jerked up by a tap of the finger, on a lever, placed below it, and allowed at once to fall, so that it will be seen that a similar curve, due to the rebound of the sphygmograph ^{weights} is produced and this is evidently the cause of the curve (b) in tracings 8 & 9. The manometer, however in 9, has produced a very marked curve (a) which corresponds in every way to the diastolic notch in an ordinary tracing. The effect of the introduction of the manometer is well seen, by comparing 2 & 3 in which Bourdon-Sander son's schema was used, a long pause being made between the opening of auricular-ventricular valve, and the closure of the aortic. In No 2 the manometer was employed, in 3 it was shut off. The other points ~~dwelt~~ brought out by this trace are dwelt upon above. In Nos 4 & 5 the sphygmograph and tube were in mercury, and the bottom of the sphygmograph was cemented to the tube. In 4 the manometer was employed, in 5 it was left out, the other conditions being the same. The effect of this, in suppressing all subsidiary vibrations, and in 4 where the manometer is in action producing a close resemblance to the down stroke of a normal pulse, is most marked. The two tracings 4 & 5 were produced by simply opening and closing the tube with the finger. From these experiments then, it is plain, the injection of fluid, into the part of the tube representing the aorta, at the

moment succeeding its utmost distension, produces a wave in all respects exactly corresponding with the dicrotic. This injection of fluid closely simulates the effect of the contraction of the aorta, acting like it in two directions, the one half aiding the centrifugal flow of the fluid; the other, being projected against the aortic valve, and by its rebound from this, producing a second or dicrotic wave. If this is the case, a tracing taken from the artificial aorta nearer the heart than ~~at~~ the point at which the injection is made, ~~should~~ should show first a rise produced by the fluid flowing towards the heart, immediately followed by a second rise due to its rebound, and this will be seen to be the case on examination of the trace 112, which was obtained on the central side of the expansion of the aorta, compressed by weights as previously described; and, what is more, the second curve, produced by this reflux, should precede in time the dicrotic notch in the artery by an amount varying with the length of tube intervening. In Vol II we have two synchronous tracings taken, the one (a), above the one (b), some distance below the distension, and the corresponding points in each marked in the usual way. It will be seen that the second or reflux wave in (a) precedes by a distinct interval, the dicrotic notch in (b) and in tracing 12, in which the

Sphygmograph, giving the tracing (C), was placed much nearer the expansion, it is plain that this interval is diminished. This goes far to show that it is this rebounding wave which produces the notch. Indeed it has been shown by Chauveau and Marey that such a centripetal wave occurs just at this period in the carotid of the horse.

Applicability of Theory to Physiological Variations

Let us now consider how this theory explains the alterations in the intensity of diastole under different conditions. These conditions are very explicitly laid down by Marey and may be thus summed up.

Conditions increasing the diastolic wave.

(a) Fast pulse (b) Elastic vessels

(c) Feeble tension in vessels

(d) Small quantity of fluid rejected at each ventricular systole. The reverse of these conditions produces decrease of the wave.

The reason why all these increase the diastolic notch appears to be one and the same - viz - that the amount of a fluid rebound depends more on the velocity of the impact than upon the mass projected. In (a) and (d) which are practically

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two ways of stating the same thing, we have a short sharp distention of the aorta, followed by sudden cessation of the expanding force. As the contractile force of elastic tissue is greater, the less the amount of previous extension we will have the fluid driven back faster, and a greater rebound produced despite the fact that the mass of fluid is less. In (b) we find a strong confirmation of the explanation advanced, the greater elasticity of the aorta necessarily producing a more powerful reflux wave. In (c), which is perhaps the most important from a clinical point of view, we have to deal with a diminished tension in the aorta, but probably not with a proportionally reduced expansion by the ventricular contraction, so that the aorta, having to contract against less resistance, will do so more rapidly and forcibly, and thus produce a rapid centripetal wave.

Application to pathological variations

It remains to us to consider how pathological conditions influence the wave. The chief of these are

- (a) Aneurisms of the Aorta
- (b) Obstructions of the Aortic orifice
- (c) Incompetency of Aortic valves
- (d) Rigidity of arteries

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In aortic aneurisms two conditions, may be more or less distinctly present. Either we may have what is practically a dilatation of the aorta, or a sac opening off it by a comparatively narrow orifice. Upon the above theory we would expect these conditions to produce opposite effects upon the diastolic wave, the first diminishing, the second ^{*1}increasing it. Mr. Marey has shown that in the first case we find diminution, if not complete extinction, of the wave. In the second case, a marked increase, as we would expect, is found ^{*2}both in a patient with this affection, and also in an imitation of the disease on the Schema.

(B) In aortic obstruction the marked absence of diastolic wave is also confirmatory of the views advanced. Here the comparative slowness of the entrance of the blood into the aorta permits of its discharge into the peripheral circulation, without ^{*3}the distention of the aorta, sufficient to give rise to a contraction of great enough extent to produce a rebound.

(C) In incompetency of the valves, on the other hand, we sometimes ^{*4}have marked diastolic wave, but, as Marey points out, this is —

*1	Circulation du sang.	p 461
*2	Marey op. cit.	458
*3	" " "	504
*4	" " "	570

accompanied by low tension, and a very sudden and high rise of the upstroke showing a great and sudden distention of the aorta; conditions which we have seen are peculiarly favourable to the production of the wave according to the explanation now offered.

(d) In rigid arteries on the other hand we have suppression of the wave - this, however, we would naturally expect on almost any theory.

Dr Galabin failed to obtain a marked diastolic trace from the aorta itself, but this, as well as some other observations by various authors, is, probably, explained by the consideration that for the same amount of fluid injected into a given length of vessel the excursion of the wall of the vessel will be less the greater its sectional area, so that a wave of fluid capable of producing a marked distention of the radial might be quite inadequate to give a motion of the aortic wall of sufficient extent to record itself.

Conclusions

To sum up it has been shown that, on general physiological grounds, it must be admitted that the contraction of the aorta, immediately succeeding its expansion by the ventricular systole, must produce a current

of blood flowing towards the heart, as well as one flowing towards the periphery and that this current impinging on the aortic valves ~~as~~ their substitute must be reflected. A simulation of this state of matters in a simple schema produces a dicrotic notch which without^{it} is wanting. Therefore, this reflux of blood towards the heart, produced by the aortic contraction, and reflected from the cardiac end of the aorta, produces the dicrotic notch. This is borne out by experiment - shewing that by a simulation of these conditions, the dicrotic wave can be produced, when, all other things being equal, it is equal absent.

In Physiology and Pathology, it explains how the variations, in intensity of the dicrotic wave, can be satisfactorily accounted for, by the theory advanced. In conclusion, it may be stated that although typical tracings have been selected to lay before you, numerous others were taken bearing out equally well the propositions advanced.

Postscript - Since writing the above I find that - Dr. Mahomed has advanced in the pages of the Medical Times

and Gazette (1873 p.490) a theory of
diastole somewhat similar to the
one first propounded but as far as
I can understand from the short-
account there given while asserting
that "the elastic recoil of the aorta
must be admitted as the prime
origin of the diastolic wave" he seems
to consider that this action is direct-
and not reflected from the cardiac
end of the aorta. Tracings 11 & 12
seem to show that this is not the
explanation.

Explanation of Tracings

- 1 Curve produced by opening and closing ^{with} the finger and thumb an elastic tube arranged so as to give a head of water of $1\frac{1}{2}$ metres, above the point of closure and to extend two metres beyond, in a nearly horizontal line ending in a smaller ext tube. The sphygmograph was placed about a metre from the point where the tube was compressed.
- 2 Burdon Sanderson's schema with the addition of a mercurial manometer of about the same diameter as the elastic tube connected by a T piece with the portion of tube representing the aorta, six inches from the part of the apparatus which imitates the aortic valve. The distal end of the manometer was closed.
- 3 The same arrangement but the manometer shut out by a strong clip.
- 4 Simple tube as in No. 1 the manometer being added as in No. 2 the sphygmograph ~~and~~

button and a portion of the elastic tube immersed in mercury. Peripheral resistance by a mass of water in a vessel (Fig. V)

5 The same but manometer shut off

6 The same conditions as No 2 and 3 the resistance considerable and the manometer off

7 The same but the manometer in action

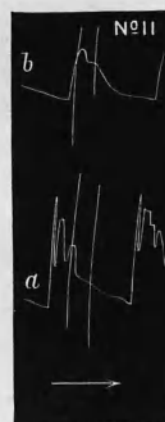
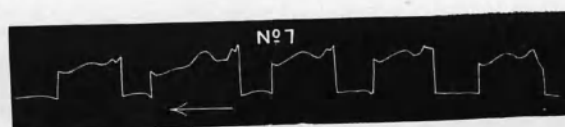
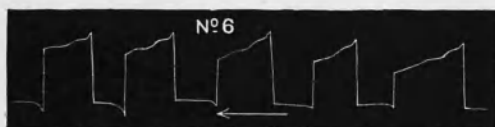
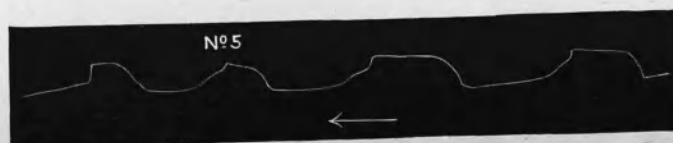
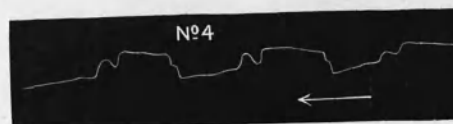
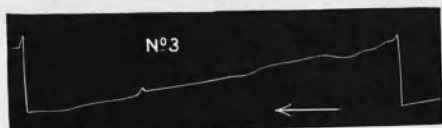
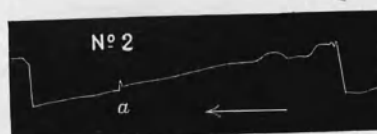
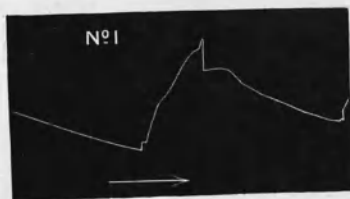
8 The tubing beyond the valve all glass except short junctions and two inches immediately beyond the manometer on which the sphygmograph was placed. The resistance as in No 4 the manometer shut off

9 The same manometer on

10 Sphygmograph ~~and~~ ^{on} partially filled tube moved by taps on a lever placed below the support for the weights

11 Synchronous tracing from each side of an india-rubber expansion (a Higginson's syringe without valves) introduced into the continuity of the tube at the part representing the aorta. An ordinary Higginson's syringe worked by the hand being used to represent the ventricle. The resistance was reduced by placing the tube of water into which the exit tube was laid below the level of the table ^{on} at which the artificial vessel lay. The expanded part was compressed by a weight of two pounds placed on the end of a flat lever giving a pressure about double that of the actual weight used. The sphygmograph giving tracing (a) being between the pump and the expansion. ~~The~~ The upper tracing (b) being taken from a point at a considerable distance from the expansion.

12 The same but trace (b) taken near the expansion.



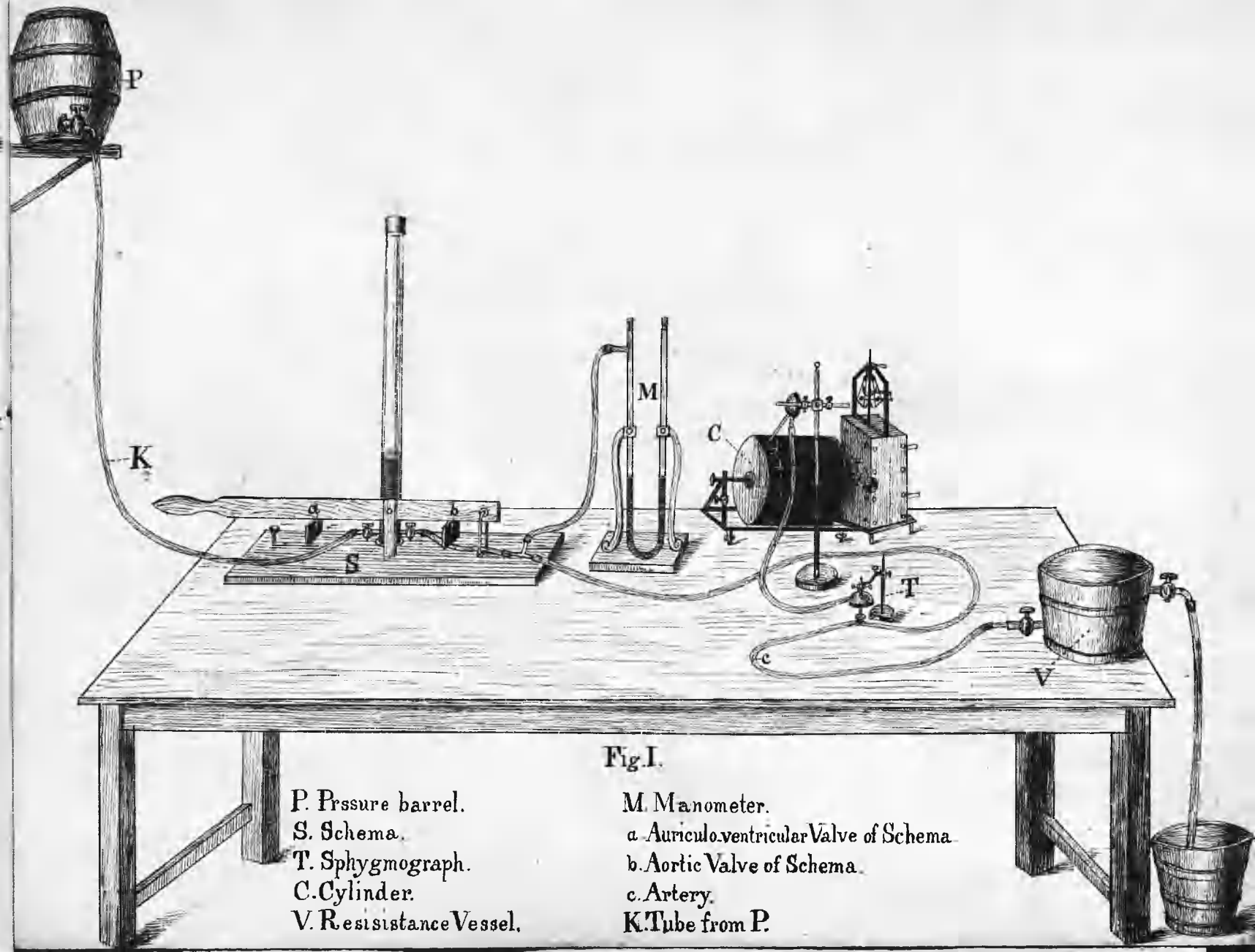


Fig. I.

P. Pressure barrel.
S. Schema.
T. Sphygmograph.
C. Cylinder.
V. Resistance Vessel.

M. Manometer.
a. Auriculo-ventricular Valve of Schema.
b. Aortic Valve of Schema.
c. Artery.
K. Tube from P.

Tracings.

No 1



No 2.



No 3



No 4



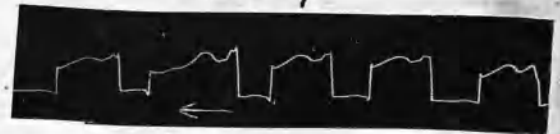
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No 6



No. 7



No. 8



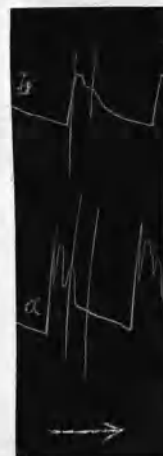
No. 9



No. 10



No. 11



No. 12



Read in the direction of the arrows.